In the Specification:

Please amend the Brief Description of the Drawings starting on page 1, line 23 to page 3, line 20 as follows. Note that the description of Figure 7 has not been amended.

Figure 1 shows an analysis of regulatory elements that govern T-cell activation in the presence of T-cell mitogens, in the presence and absence of immunomodulatory drugs.

Plotted is the activation profile of 16 difference combinations of mitogens (see table one) in the absence of immunomodulatory drugs ~~(red)~~(speckled circles), the presence of 100 nM cyclosporine A ~~(yellow)~~(open circles) and with 10 uM SB203589, an anti-inflammatory mitogen activated kinase inhibitor ~~(blue)~~(filled circles).

Figure 2 shows an analysis by hierarchical clustering.

The data from Figure 1 were re-analyzed by hierarchical clustering. On the left is shown a heat diagram that indicates the clustering of the activation profiles of the mitogen and drug combinations (rows) according to how they activate the eight different promoter elements or "transcriptional targets" (columns). The ~~color code~~ intensity indicates the % maximum fold activation x100. On the right is a dendrogram that represents the similarities and differences between the promoter elements based on how they responded to the mitogen/drug combinations as illustrated on the right.

Figure 3 shows an analysis by non-supervised hierarchical clustering.

This analysis distinguishes and groups transcriptional targets by the drug sensitivity of mitogen response profiles. Analysis of the data in Figure 3 by self-organizing maps (SOM) separates the 8 promoter elements into 4 distinct classes. ~~(Top)~~ In Fig. 3A, ~~E~~each corner of the square indicates a separate class containing promoter elements that respond similarly. The size of the circles indicate the size of the class. ~~(Bottom) The bottom plot~~ Fig. 3B shows an average profile or "centroid" for each class.

Figure 4 shows Principal Components Analysis (PCA) of multiple drug profiles.

The conditions outlined in Table I were analyzed by principle components analysis, which reveals major outliers in the presence of IGF-1 (the four ~~blue data~~ points represented by speckled circles, individually labeled).

Figure 5 shows Principal Component Analysis of vector differences between mitogen profiles in the absence and presence of drug.

This analysis reveals significant contrasts and similarities of drug targeting. The mitogen activation profiles under control conditions show in ~~red~~ Figure 4 were used to calculate subtraction vectors for each profile in the presence of a drug treatment. These vectors were then analyzed by PCA to show similarities and differences in targeting.

Figure 6 shows a "widescreen" analysis of transcriptional activation profiles from high throughput transfections.

This analysis reveals clear patters of drug, mitogen and hormone action. **Fig. 6** shows a heat diagram from 3072 transfection under the conditions indicated. Sixteen different combinations of mitogens (see table one) were assayed for their ability to stimulate 8 promoter elements (columns) in the presence and absence of different immuno-modulatory agents. The data are represented in % maximum activation x 100. ~~Color~~ Ceode on the right indicates 0% to 170% in a ~~color~~ gradient from ~~blue to red~~ solid to speckled. (~~Pink~~Dark speckled indicating 100% maximal stimulation in the absence of modifying agents).

For example, the mitogen Wortmannin strongly stimulates AP-1 and CRE and, to a lesser extent, UAS/p300FL; IGF-1 strongly stimulates CD28RE/AP1 and other promoters shown; and CSA strongly stimulates AP-1 and, to a lesser extent, CRE and UAS/p300 N-term.

Figures 8A and 8B show an analysis of mitogen profiles in "proteomic space."

Figure 8A shows a PCA analysis of the manner in which the mitogen profile of the levels of phospho-retinoblastoma protein, phospho-AKT kinase, PCNA protein, phospho-jun protein, and total phospho-tyrosine levels are influenced in T-cells in the presence of cyclosporine A (control profile in ~~red~~ speckled circles, 100 nM cyclosporine treated in ~~green~~ open circles). **Figure 8B** shows a PCA analysis of how the profile changes with treatment in the presence of 10 nM TGFbeta (control in ~~red~~ speckled squares, TGF beta in ~~green~~ open squares).

Page 43, line 30 "activation. The labeled outliers ~~(blue)~~ in Figure 4 indicate that the combined presence of"

Page 44, lines 4-6 "PI3 kinase inhibitor, wortmannin and IGF-1. Forskolin ~~(brown)~~ shows a targeting profile that is completely different from the other conditions, whereas IGF ~~(blue)~~ and wortmannin ~~(yellow)~~ appear to target the same pathway. Indeed, it is known that IGF-1"